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## Longitudinal pathways between mental health difficulties and academic performance during middle childhood and early adolescence

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**Longitudinal pathways between mental health difficulties and academic performance  
during middle childhood and early adolescence**

**Abstract**

There is a growing appreciation that child functioning in different domains, levels, or systems are interrelated over time. Here we investigate links between internalizing symptoms, externalizing problems and academic attainment during middle childhood and early adolescence, drawing on two large datasets (child: mean age 8.7 at enrolment,  $n=5,878$ ; adolescent: mean age 11.7,  $n=6,388$ ). Using a two-year cross-lag design, we test three hypotheses—adjustment erosion, academic incompetence, and shared risk—while also examining the moderating influence of gender. Multi-level structural equation models provided consistent evidence of the deleterious effect of externalizing problems on later academic achievement in both cohorts, supporting the adjustment-erosion hypothesis. Evidence supporting the academic incompetence hypothesis was restricted to the middle childhood cohort, revealing links between early academic failure and later internalizing symptoms. In both cohorts, inclusion of shared risk variables improved model fit and rendered some previously established cross-lag pathways non-significant. Implications of these findings are discussed, and study strengths and limitations noted.

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## **Introduction**

A longstanding and primary focus of human development research concerns whether and how functioning in one domain at one point in time predicts and presumably affects functioning in other domains at a later point in time. Investigation of such cross-time/cross-domain links is often referred to in terms of “developmental cascades” (Masten & Cicchetti, 2010).

In this report we draw on the developmental cascade literature to develop a two-year cross-lag model that examines, longitudinally, inter-relationships between internalizing symptoms, externalizing problems and academic performance. Using two large child- and adolescent-data sets, we test three alternative hypotheses pertaining to how these constructs relate to one another over a two-year period—adjustment erosion, academic incompetence, and shared risk (Masten et al., 2005; Moilanen, Shaw, & Maxwell, 2010), each of which is defined below.

Exploring longitudinal pathways between mental health and academic performance in children and adolescents is an important focus in research on developmental cascades (e.g., Ansary & Luthar, 2009; Bennett, Brown, Boyle, Racine, & Offord, 2003; McCarty et al., 2008; Moilanen, et al., 2010). Given the apparent influence of functioning in these domains when predicting diverse aspects of adult development (e.g., Healey, Knapp, & Farrington, 2004), this is perhaps unsurprising. Extensive evidence indicates that mental health problems identified in childhood and adolescence constitute a major ‘disease burden’ in terms of their implications for physical health and mortality, as well as future economic and social functioning (Patel, Flisher, Hetrick, & McGorry, 2007). Academic achievement also has long-term implications, not just for future academic success and employment, but also for depression, alcoholism, and violent behaviour (Ek, Sovio, Remes, & Järvelin, 2005; Kosterman, Graham, Hawkins, Catalano, & Herrenkohl, 2001; Pelkonen, Marttunen, & Aro,

2003; Windle, Mun, & Windle, 2005). Developing knowledge and understanding of the nature, magnitude and stability of such relationships over time can contribute to developmental theory, which can inform prevention and intervention efforts.

Three models have been proffered to explain how externalizing problems, internalizing symptoms and academic competence are linked across time. The *adjustment-erosion* model stipulates that internalizing and/or externalizing difficulties lead to later academic difficulties (Moilanen, et al., 2010). For example, aggressive behaviours can present a barrier to learning because of their adverse effects on peer acceptance and relationships with teachers. Similarly, emotional distress may impede academic progress by altering cognitive functions related to learning, thereby reducing participation in the classroom (Moilanen et al., 2010).

The relationship between internalizing symptoms and externalizing difficulties is also part of the adjustment-erosion model, in that early externalizing difficulties are presumed to foster later internalizing symptoms. This is because behavioural problems are hypothesized to disrupt interpersonal relationships, which subsequently affects emotional distress. By contrast, initial internalizing symptoms are assumed to be *inversely* related to later externalizing problems because the self-isolation and withdrawal associated with the former are assumed to reduce the risk of future disruptive behaviour (Masten et al., 2005).

Some evidence provides support for the adjustment-erosion model. In the United States (US), Moilanen and associates (2010) found that externalizing difficulties predicted low academic competence and high internalizing symptoms over a six-year period from middle childhood to early adolescence. Masten et al. (2005) discerned similar results from middle childhood to adulthood, while also observing that early internalizing symptoms proved “protective” with respect to future externalizing problems. Also consistent with the adjustment-erosion model are Obradovic, Burt and Masten’s (2010) data indicating that

## LONGITUDINAL PATHWAYS BETWEEN MENTAL HEALTH AND ACADEMIC PERFORMANCE

internalizing symptoms in childhood forecast reduced academic competence in adolescence.

However, Duncan et al. (2007) detected no significant links between internalizing or externalizing problems and later academic achievement. Similarly, the anticipated relations between internalizing symptoms and externalizing problems have not consistently emerged; Moilanen et al. (2010) failed to find evidence of the former predicting the latter.

The *academic-incompetence* model reverses the cascading processes central to the adjustment-erosion model, stipulating that problems relating to academic competence initiate or exacerbate existing internalizing and/or externalizing difficulties (Moilanen et al., 2010). From this perspective, low academic achievement is presumed to engender frustration, disaffection and/or lead to educational placements that increase exposure to deviant peers, increasing the likelihood of disruptive behaviour. In terms of internalizing, experience of academic failure may lead to feelings of worthlessness or low self-esteem (Maughan, Rowe, Loeber & Stouthamer-Loeber, 2003).

Empirical support for the academic-incompetence model can be found in a number of studies. In a Canadian sample, Bennett et al. (2003) observed that limited reading achievement at school entry forecast more conduct problems 30 months later. Similarly, Vaillancourt and associates (2013) found that a low grade point average in Grades 5–7 predicted increasing externalizing symptoms a year later. In relation to internalizing symptoms, McCarty et al. (2008) reported that adolescent school failure predisposed American females to depressive symptoms in early adulthood. Finally, results of Moilanen et al.'s (2010) study chronicled adverse effects of low academic competence on later internalizing *and* externalizing problems, even if not across all hypothesized paths. For example, lower academic competence predicted higher internalizing symptoms in middle childhood but not early adolescence.

## LONGITUDINAL PATHWAYS BETWEEN MENTAL HEALTH AND ACADEMIC PERFORMANCE

The third and final model, referred to as the *shared-risk* hypothesis (also known as “common cause”; Bornstein, Hahn, & Suwalsky, 2013b), stipulates that the effects central to the first two models are actually explained by “third variables” affecting multiple, interrelated domains of development (Moilanen, et al., 2010). Typically these are operationalized as risk markers, the most common being intellectual ability, parenting quality, and deprivation or socio-economic status (Masten et al., 2005). Taking the latter as an example, socio-economic disadvantage is known to predict both social-emotional difficulties and poor academic attainment (Bradley & Corwyn, 2002). The increased risk associated with poverty is attributed to a lack of access to resources that more wealthy families can afford, in addition to exposure to stressors, such as poor housing quality and a lack of structure (Bradley & Corwyn, 2002; Hetzner, Johnson, & Brooks-Gunn, 2010).

The standard approach to assessing the influence of shared risk variables in developmental-cascade research has been to include them as co-variables and subsequently contrast models. Evidence to date indicates that although model fit typically benefits from the inclusion of shared-risk variables, most previously established cascade pathways are generally unaffected (Bornstein, Hahn & Suwalsky, 2013b; Masten et al., 2005; Moilanen et al, 2010).

### **The Current Study**

The aim of the current study was to extend the research on longitudinal associations between internalizing symptoms, externalizing difficulties, and academic achievement using large, representative samples of children and adolescents in England. The dataset used in this analysis was procured during a major longitudinal research project on school-based mental health (Deighton et al., 2013; Vostanis, Humphrey, Fitzgerald, Deighton, & Wolpert, 2013; Wolpert et al., 2011; Wolpert, Humphrey, Belsky, & Deighton, 2013).

[INSERT FIGURE 1 ABOUT HERE]

The current study draws on this large database to address several limitations and inconsistencies in the extant evidence base. First, sample sizes in many studies have been relatively modest. This reduces the sensitivity of statistical tests to account for nesting in the data, or estimate correlated errors, to detect non-zero paths (Masten et al., 2005) and limits opportunities for including additional parameters (Bornstein, et al., 2013a). Additionally, there are issues with the representativeness and comparability of samples used in existing work. It is difficult to compare the findings of, for example, Ansary and Luthar's (2009) study of affluent adolescents with those of Moilanen et al. (2010), who studied an economically disadvantaged sample. The same applies for Masten et al.'s (2005) models evaluating predictive relations across 20 years, with up to 10-year periods between measurements, with those addressing time lags of just one or two years (e.g., Moilanen et al., 2010). Herein we examine time lags of two years, based upon the design of the larger study from which the data for this report were drawn.

Second, the majority of the research investigating cascading associations has been carried out in the US. Cultural transferability of findings cannot be assumed because of the primacy of cultural context in the developmental eco-system (Bronfenbrenner, 2005). For example, there are differences between the US and UK in terms of the age at which out-of-home childcare begins, typical class size and the average number of educational transitions. To date there has only been one UK-based study of the developmental cascades between internalizing symptoms, externalizing problems and academic attainment. Panayiotou and Humphrey (2017) found support for both the adjustment erosion and academic incompetence hypotheses, but the pathways observed were gender-specific (the former only evident for

boys, the latter only for girls). They also found that the influence of shared risk was meagre, improving model fit but substantively affecting only one previously established pathway. However, these authors' analyses were restricted to middle childhood, limiting their ability to assess the extent to which identified pathways intensify or attenuate through the course of development. Furthermore, the use of teacher informant-report mental health data raises concerns about the accuracy with which internalizing symptoms are recorded (in particular, teachers may under-report difficulties in this domain; Kolko & Kazdin, 1993). The current study addresses these acknowledged limitations, using data covering two distinct developmental periods (middle childhood and early adolescence) and self-report survey methods (which are optimal for the assessment of internalizing symptoms; Whitcomb & Merrell, 2013).

Third, the current study also provides an opportunity to clarify the role of internalizing difficulties in the developmental process (Masten & Cicchetti, 2010; Moilanen, et al., 2010). In particular, the exact nature of the association between internalizing and externalizing difficulties over time remains elusive; whereas some work chronicles positive associations, other research documents negative ones (Masten et al., 2005).

Fourth, we evaluate whether the longitudinal pathways vary by sex. Mental health problems vary as a function of gender, with higher levels of internalizing symptoms among girls and greater externalizing problems among boys, though these gender differences become more and less pronounced, respectively, as children develop (Green, McGinnity, Meltzer, Ford, & Goodman, 2005). Sex differences are also evident in academic attainment, in both primary and secondary school contexts, with girls out-performing boys (DfE, 2011). There is some evidence, however, of change in this gender difference with age (Riglin, Petrides, Frederickson, & Rice, 2013). For example, McCarty et al. (2008) found evidence that academic performance affected later depressive symptoms among adolescent girls, but



not boys. Similarly, Klostermann, Connell and Stormshak's (2016) work indicated that externalizing problems appeared to precede internalizing symptoms for boys in early adolescence, whereas the reverse was true for girls.

Fifth, in relation to the shared-risk model, data available in the current study enabled us to consider an alternative risk marker that has been relatively neglected in the extant literature. Specifically, being identified as having special educational needs (SEN) has been found to co-vary with both academic achievement (DfE, 2011a) and mental health difficulties (Green, et al., 2005). Children and adolescents with SEN are likely to be victims of bullying (Fink, Deighton, Humphrey, & Wolpert, 2015), experience lower peer acceptance, and have fewer friends (Pijl, Frostad, & Flem, 2008) than their peers. We also included socio-economic deprivation as a well-known correlate of both mental health problems (Caspi, Taylor, Moffitt & Plomin, 2000) and academic attainment (Sirin, 2005).

In summary, the primary aim of the research reported was to illuminate longitudinal pathways linking internalizing symptoms, externalizing problems and academic attainment consistent with the models of adjustment erosion and academic incompetence, as displayed in Figure 1. We also aimed to examine the role of two potential “shared-risk” factors that could influence both mental health and academic attainment, socio-economic deprivation and SEN. A secondary goal was to consider whether these processes were moderated by gender. Although the work presented herein is informed by the developmental cascade literature, which typically incorporates at least three time points, only data from two time points were available on academic achievement.

Our analyses explore the relationships between mental health and academic attainment across two distinct developmental phases (middle childhood, age 8 to 11; and early adolescence, age 11 to 14) and school contexts (primary school and secondary school). There are several reasons we might expect to see variation in cascade pathways between

mental health and academic attainment across these developmental epochs. The primary- and secondary-school contexts in England are very distinct. Relative to primary schools, secondary schools are much larger and place greater emphasis on relative ability and competition and rules of behaviour. Children also experience less personal relations with teachers than in primary schools (Humphrey & Ainscow, 2006). Finally, early adolescence is a period during which major biological, cognitive and social changes occur that may have implications for mental health (Arnett, 1999) and, indeed, research documents increased prevalence of both internalizing and externalizing problems during this period (Green et al., 2005).

Based on the preceding theory and evidence, we made several predictions. First, consistent with the adjustment-erosion model, we predicted that greater initial externalizing difficulties and internalizing symptoms would forecast poorer later academic attainment. Additionally, we hypothesized that more early externalizing problems would forecast more internalizing symptoms but that more initial internalizing symptoms would forecast fewer externalizing difficulties. Second, in line with the academic-incompetence hypothesis, we predicted that early academic attainment would be inversely related to later internalizing and externalizing problems. Third, we expected that the just-delineated cross-time associations would be evident even after accounting for shared risk. Finally, we hypothesized that these associations would vary as a function of both gender and developmental period, but did not specify in advance exactly how, given the nascent status of the evidence base. It should be noted that the modelling employed always involved early functioning in one domain of functioning predicting change from earlier-to-later functioning in another domain, in that the later outcome was adjusted for its earlier measurement.

## **Method**

### *Participants*

The overall sample consisted of 12,266 children (5,878 primary school and 6,388 secondary school) who were participants in one of two cohorts of a longitudinal research project on school-based mental health. The primary-school cohort included children aged eight to nine (mean age 8.7, SD 0.30) at the outset of the study. The secondary-school cohort included adolescents aged 11 to 12 (mean age 11.7, SD 0.30). The sample included any child from 157 primary schools and 41 secondary schools who completed a survey at at least one time point and for whom shared-risk data were available.

Of the primary-school sample, 51.2% were male and 73% were classified as ‘White’. These proportions closely mirror the composition of the English primary-school population, albeit with a higher proportion of children from ethnic minorities (national proportions: 78.5% White, 51.07% male; DfE, 2010). Children eligible for free school meals (FSM) constituted 21.9% of the sample, somewhat higher than the national average of 17.3% for primary-school children (DfE, 2010). Average academic attainment was derived from the most recent national assessment scores for English, Maths and Science. In the current sample it was 14.68, which is lower than the national average of 15.30 (DfE, 2010).

Of the secondary school sample, 46.4% were male and 78% were classified as ‘White’. These proportions are similar to the composition of the secondary-school population of England (national proportions: 81.2% White, 50.41% male; DfE, 2010). Children eligible for FSM constituted 18.6% of the sample, higher than the national average of 14.2% for secondary-school children (DfE, 2010). Average academic attainment was 27.3 (for further details, see academic attainment section in ‘measures’), which is similar to the national average (DfE, 2010).

### ***Procedure***

Children completed a self-report questionnaire in autumn 2008 (T1) and autumn 2010 (T2) using a secure online website. Class teachers facilitated online, whole-class sessions for completing the questionnaire, having been provided with a standardized instruction sheet to read aloud. The sheet outlined what the questionnaire was about, the confidentiality of answers, and each child's right to decline participation. The online survey system was designed to be easy to read and child friendly.

### ***Measures***

*Internalizing symptoms and externalizing problems.* Internalizing symptoms and externalizing problems were measured using the *Me and My Feelings* (M&MF, formerly *Me and My School*, M&MS) scale (Deighton et al., 2013; Patalay, Deighton, Fonagy, Vostanis, & Wolpert, 2014). The M&MF scale contains 16 items. Each item consists of a statement (e.g., "I worry a lot" [internalizing], "I lose my temper" [externalizing] – see Appendix 1 for full list), combined with a three-point response scale (never, sometimes, always). The Flesch-Kincaid Grade Level score for the measure is c.0.6, indicating that the items are simple enough to be read and understood by an average six-year-old child.

Psychometric analyses indicate that the M&MF has good content validity; strong internal consistency (based on confirmatory factor analysis and Cronbach's Alpha coefficients of >0.7, Deighton et al., 2013) (based on correlations with the Strengths and Difficulties Questionnaire – Goodman, 1997) (Deighton et al., 2013).

*Academic attainment.* Academic attainment was measured using end of Key Stage (KS) assessment data derived from the National Pupil Database (NPD) held by the English government's Department for Education (DfE). Compulsory education in England is divided into Key Stages: Year 1 and 2 (ages 5–7; KS1), Years 3, 4, 5 and 6 (ages 7–11; KS2), Years

## LONGITUDINAL PATHWAYS BETWEEN MENTAL HEALTH AND ACADEMIC PERFORMANCE

7, 8 and 9 (ages 11–14; KS3), and Years 10 and 11 (ages 14–16; KS4). Assessments occur at the end of these key stages. Each provides data corresponding to attainment in core curriculum subjects (e.g., English, Maths, and Science). For the primary school cohort academic attainment was measured using teacher assessment data at the end of KS1 (Time 1) and standardized test data at the end of KS2 (Time 2), both of which produce point scores for English, Maths, and Science. For the secondary school cohort, academic attainment was captured using standardized test data at the end of KS2 (Time 1) and teacher assessment data at the end of KS3 (Time 2). These data are scored such that higher scores reflect higher academic attainment.

*Special Educational Needs (SEN).* SEN status was based on data collected centrally by the DfE via the NPD. These data provide information about the nature of educational provision for a given child on an ordinal scale as shown in Table 1.

[INSERT TABLE 1 HERE]

*Socio-economic deprivation.* Socio-economic deprivation was assessed using children's Income Deprivation Affecting Children Index (IDACI) scores, which are also stored in the NPD. IDACI is derived from the deprivation ranking of the neighbourhood (or “super output area”) within which a child lives (McLennan et al., 2011). The score represents the proportion of children under 16 in that area who live in a low-income household. It is a metric between 0 and 1, with higher scores reflecting higher levels of deprivation.

### **Analysis**

## LONGITUDINAL PATHWAYS BETWEEN MENTAL HEALTH AND ACADEMIC PERFORMANCE

Cross-lag models were tested by multi-level structural equation models using MPlus version 7 (Muthén & Muthén, 2015; all models with WLSM estimator);<sup>1</sup> schools served as a clustering variable, with children ‘nested’ within schools. Analyses were carried out separately for primary and secondary school cohorts. Factor loadings and thresholds for internalizing and externalizing scores were constrained to be the same across time points, assuming strong factorial invariance. To additionally examine whether gender differences existed, we ran multi-group (gender) structural equation models controlling for clustering by schools using cluster-robust standard errors, which revealed no significant difference between boys and girls in either primary or secondary school samples (see supplementary material 1). Hence, the final models within the two school cohorts were not disaggregated by gender.

Model fit was considered to be acceptable if Comparative Fit Index (CFI) and Tucker-Lewis Index (TLI) were above .95 and Root Mean Square Error of Approximation (RMSEA) was below .05 (Bollen & Curran, 2006). Two versions of the models were tested. The first version accounted for (a) temporal stability in internalizing symptoms, externalizing problems, and academic attainment; (b) concurrent correlations between these variables; and (c) cascade pathways across domains over time (see Figure 1) (Bornstein, et al., 2013a). In this model, shared-risk variables (i.e., deprivation, SEN) were included, but all pathways from these variables were fixed to zero (see Supplementary material 2 for further details). In the second version of the models, pathways involving shared-risk variables were freed, allowing deprivation and SEN to predict internalizing symptoms, externalizing problems, and academic attainment at each time point. Improvement in model fit between basic and shared-risk models was tested using the Satorra-Bentler scaled chi-square difference test (Satorra, 2000). Only the models with shared-risk variable paths freed are presented in the figures.

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<sup>1</sup> Following Vaillancourt et al. (2013), we report the range of (T1) intra-class correlations (ICCs) for items making up the study variables. The primary school sample ICC ranges were .12–.18 for indicators of academic attainment, .00–.03 for indicators of internalizing symptoms, and .02–.07 for indicators of externalizing problems. In the secondary school sample the ranges were .05–.21, .01–.03 and .01–.04 respectively.

## LONGITUDINAL PATHWAYS BETWEEN MENTAL HEALTH AND ACADEMIC PERFORMANCE

Given the large sample size, pathways in these models are only reported as significant at  $p < .01$ .

### Results

Figures 2 and 3 depict the results for the primary- and secondary-school samples. All models demonstrated fair to good fit to the data ( $CFI$  and  $TLI \geq .95$ , and  $RMSEA \leq .05$ ). In all cases, within-construct temporal stability was strong, particularly for academic attainment ( $\beta > .86$ ,  $p < .001$ ).

**Primary-school results.** The model for primary school children with shared risk variables fixed to zero are (referred to as the ‘basic model’) revealed three significant longitudinal pathways. The first two involved low academic attainment (T1) predicting both increased internalizing symptoms (T2) ( $\beta = -.08$ ,  $p < .001$ ) and increased externalizing problems ( $\beta = -.08$ ,  $p < .001$ ). There was also a significant pathway from T1 externalizing problems and later lower academic attainment (T2) ( $\beta = -.07$ ,  $p < .001$ ).

In the model that included shared-risk variables, the pathways between T1 externalizing and later achievement and the pathway between T1 academic attainment later internalizing symptoms remained ( $\beta = -.07$ ,  $p < .001$ ; and  $\beta = -.06$ ,  $p < .001$  respectively). However, the pathway from T1 academic attainment to later externalizing problems was no longer significant (Figure 2). Including shared risk factors also resulted in a significant improvement in the overall model fit ( $TRd = 2394.76$ ,  $p < .001$ ).

[INSERT FIGURE 2 HERE]

**Secondary-school results.** The initial basic model for the secondary school sample, with shared risk variables fixed to zero, revealed two significant negative cascade pathways,

## LONGITUDINAL PATHWAYS BETWEEN MENTAL HEALTH AND ACADEMIC PERFORMANCE

one from internalizing symptoms to later academic attainment ( $\beta = -.04$ ,  $p < .01$ ) and another from externalizing problems to later academic attainment ( $\beta = -.11$ ,  $p < .001$ ). These indicated that higher levels of internalizing symptoms and externalizing problems both led to subsequent reductions in academic attainment.

Freeing the shared-risk pathways improved the model fit significantly (Figure 3;  $\text{TRd} = 694.52$ ,  $p < .01$ ). However, pathways remained similar to those observed in the basic model, meaning that pathways from externalizing problems (T1) and internalizing symptoms (T1) to academic attainment (T2) remained ( $\beta = -.10$ ,  $p < .001$ ; and  $\beta = -.04$ ,  $p < .01$  respectively).

[INSERT FIGURE 3 HERE]

### ***Summary***

In summary, multi-group analysis did not provide support for difference in models between boys and girls, therefore, analysis were carried out for combined samples of boys and girls. While multiple models were examined, once shared-risk variables were included, support emerged predominantly for the adjustment-erosion hypothesis. Longitudinal pathways proved significant for primary and secondary school age groups when linking initial externalizing problems with later academic attainment. In addition, in the secondary school sample a significant pathway from initial internalizing symptoms to later academic attainment was also detected. There was also some limited support for the academic incompetence model; in the initial primary school model, pathways were observed from initial academic attainment to later internalizing symptoms and externalizing problems. However, after accounting for shared risk variables, only the pathway from academic attainment to later internalizing symptoms remained.

### **Discussion**



## LONGITUDINAL PATHWAYS BETWEEN MENTAL HEALTH AND ACADEMIC PERFORMANCE

The current study evaluated longitudinal pathways linking mental health difficulties and academic attainment highlighted by alternative theoretical models. We drew on a large, nationally representative dataset that afforded important analytical advancements relative to much prior developmental cascade research (e.g., the estimation of latent variables, while also accounting for nesting in the data), thereby enhancing analytical rigour, while focusing separately on middle childhood and adolescence and examining potential differences between boys and girls. In contrast with Panayiotou and Humphrey (2017), multi-group analysis did not find evidence for gender differences for either age group.

Support emerged principally for the adjustment-erosion model. Recall that *adjustment-erosion* stipulates that internalizing and/or externalizing difficulties lead to later academic difficulties, whereas the *academic-incompetence* model posits that problems relating to academic competence feed forward to promote or exacerbate internalizing and/or externalizing difficulties. The *shared-risk* model, stipulates that the kinds of cascading effects central to the other models are explained by “third variables”.

The adjustment-erosion model received the most consistent empirical support. Externalizing problems appeared to undermine later academic achievement, even after accounting for shared-risk factors reflecting deprivation and SEN. These findings resonate strongly with those reported by others studying these same developmental periods (e.g., Chen, 2010; Moilanen et al., 2010), as well as results of research covering much lengthier periods of time (e.g., Masten et al., 2005). Although the co-efficient values for pathways from externalizing problems to later attainment were somewhat weaker in magnitude than those found previously, this may be explained by the fact that our structural equation models were multi-level, enabling us to account for nesting in the study variables. Some evidence also emerged consistent with the adjustment-erosion expectation that early internalizing

symptoms would undermine later academic attainment, albeit only for the secondary school sample

Also noteworthy is that the findings presented contrast markedly with those reported by Duncan et al. (2007). Such discrepancies may be partly explained by differences in age ranges, countries in which the studies were carried out and research designs, but also by methodological shortcomings, such as a lack of consideration of missing data in the Duncan et al. (2007) study. Subsequent studies have noted that addressing missing data through imputation does reveal associations between internalizing and externalizing problems and later academic competence (Romano, Babchishin, Pagani, & Kohen, 2010).

In terms of prevention, our results speak to the salience of externalizing problems as a barrier to concurrent and future opportunities for learning and participation in school. Our findings suggest that effects exist for children in primary and secondary schools. Hence, this research buttresses calls for earlier intervention (e.g., Allen, 2011) that provides an integrated approach as opposed to a “program for every problem” (Domitrovich et al., 2010). In other words, interventions that address problem behaviour could be regarded as academic-enhancement as well as behaviour-enhancement programs.

Our findings provided some indication that internalizing symptoms do predict later academic attainment, but only for the adolescent sample. Previous research has been equivocal about this relationship. For example, Masten et al., (2005) found no such pathway – something the authors speculated may have been due to internalizing symptoms having a shorter “window of influence” on attainment than they could detect with the 7 to 10 year lags in their own measures. The present study’s short lags between measures, therefore, allowed for detection of these shorter term influences. However, the magnitude of the pathway detected was small and the finding was not replicated with the primary school sample. Therefore, like Masten et al. (2005), we are left to wonder whether there may be

differential academic cascade effects for children and young people whose symptoms place them in the clinical range for internalizing symptoms, which is something we did not examine.

Evidence presented herein also provides some limited evidence for the academic-incompetence model, but only in relation to internalizing symptoms. Specifically, for primary school children, academic attainment was inversely related to later internalizing symptoms, even after accounting for shared-risk variables; however, the same pathway was not present for adolescents. This finding is consistent with previous evidence of the effect of low academic competence on later internalizing symptoms specifically in middle childhood (Moilanen, et al., 2010; Panayiotou & Humphrey (2017)), which has highlighted the potential deleterious effects of academic failure on mental health (McCarty, et al., 2008;). Explanations for the effect being observed for this specific age group include the potential impact of parent and teacher feedback around poor attainment on mood and self-concept, especially leading up to transition from primary to secondary school settings (Moilanen et al., 2010). However, this relationship and the processes underpinning it warrant further exploration. The only pathway identified was between early academic attainment and later externalizing problems for primary-school children; though this pathway was reduced to insignificance once shared risks were taken into account.

The current study provided limited support for the shared-risk hypothesis. Despite consistently improving overall model fit, the inclusion of SEN and deprivation indices rendered only one of the initial pathways non-significant, as indicated above. Thus, consistent with other researchers (e.g., Masten et al., 2005; Moilanen, et al., 2010), our conclusions regarding shared risk must remain guarded and tentative. While factors such as deprivation and SEN are salient risk markers in relation to mental health and academic attainment in childhood and adolescence, their influence on cascade effects in development

may be less powerful than initially thought; they also appear to vary by child gender.

Moreover, there is limited availability in the current study of other shared-risk factors that previous studies have acknowledged to be important, such as parenting quality.

Beyond the need to expand the list of shared-risk variables considered, there are several limitations inherent in our research design. The time lags between measurements were relatively small. In one sense this is an advantage; it helped to clarify a question raised regarding cascade effects with shorter time windows of influence (Masten et al., 2005) and enabled us to model pathways within—as opposed to across—developmental periods. However, it undoubtedly led to extremely high temporal stability, particularly in the case of academic attainment. This had (attenuating) implications for our ability to identify meaningful cross-lag cascade effects. However, such stability is perhaps to be expected, and indeed has been a feature of longitudinal research involving much longer measurement intervals than our own (e.g., Vaillancourt et al., 2013). It was also not possible to compare primary and secondary schools samples in the same model because of the different academic attainment variables for the two age groups. The final limitation is our inability to examine predictive relations across more than two times of measurement, given design of the larger project from which this report derives.

### **Conclusion**

The current study contributes to a growing literature on longitudinal associations between internalizing symptoms, externalizing problems and academic attainment in childhood and adolescence. Our analyses illuminate how functioning in these different domains are developmentally related within two different developmental periods after taking into consideration important third-variable risk factors. Our conclusions are strengthened by

## LONGITUDINAL PATHWAYS BETWEEN MENTAL HEALTH AND ACADEMIC PERFORMANCE

the use of very large samples and the use of analytical techniques that offer significantly greater rigour than has been the case in many prior studies.

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## LONGITUDINAL PATHWAYS BETWEEN MENTAL HEALTH AND ACADEMIC PERFORMANCE

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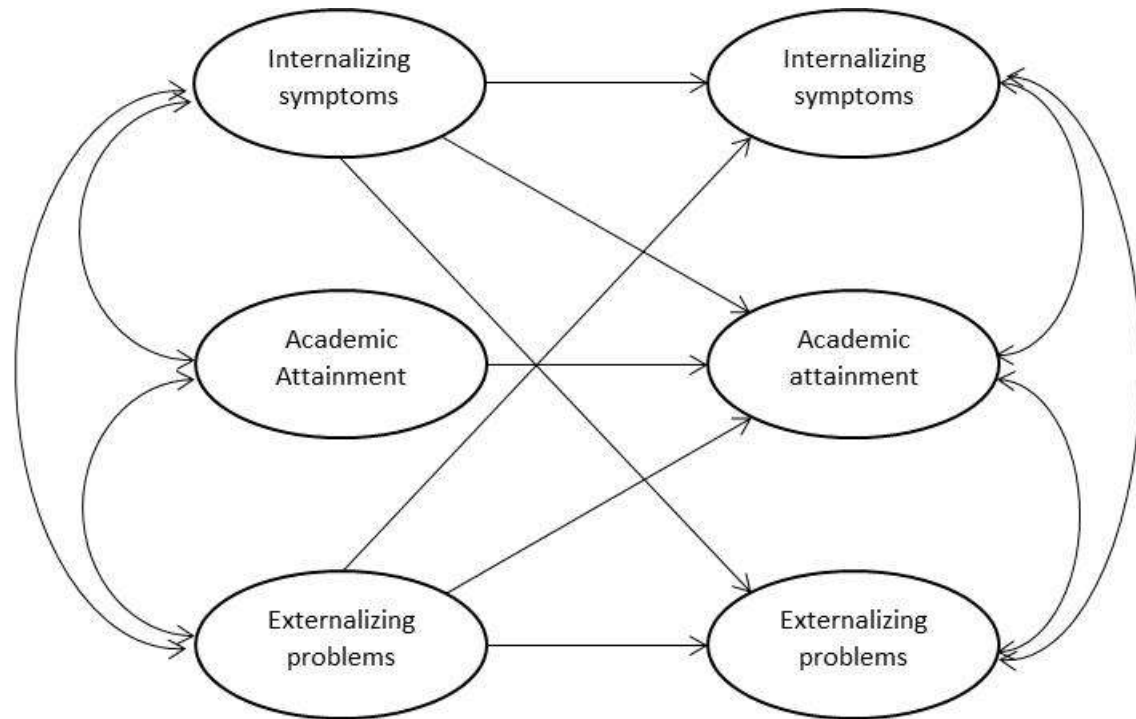
# LONGITUDINAL PATHWAYS BETWEEN MENTAL HEALTH AND ACADEMIC PERFORMANCE

Table 1

*Special Educational Needs (SEN) status categories and associated scoring*

Category	Description	Score
No SEN	No additional needs recorded	0
School Action	Identified SEN are met via adjustments to normal teaching practice	1
School Action Plus	Additional support from an external professional (e.g., educational psychologist, speech and language therapist) is required	2
Statement of SEN	A legal document which follows multi-professional assessment and outlines the nature of a child's needs and the resources required to meet them effectively	3

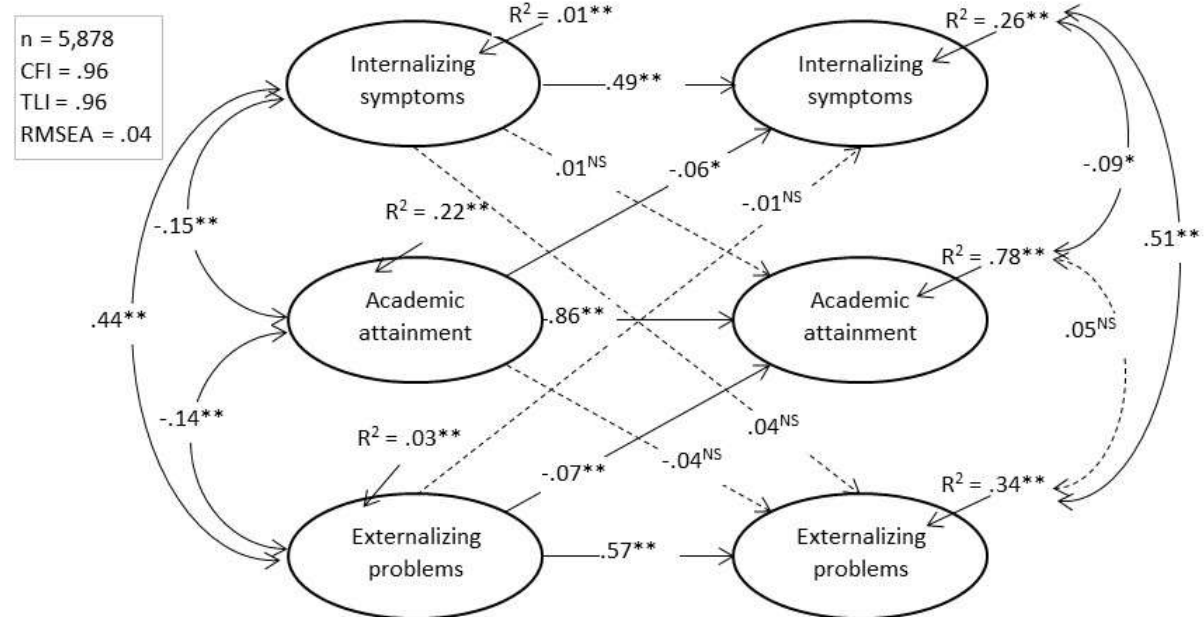
**Figure 1. Cascade models examining the longitudinal relationships  
between internalizing symptoms, externalizing problems and academic  
attainment.**



# LONGITUDINAL PATHWAYS BETWEEN MENTAL HEALTH AND ACADEMIC PERFORMANCE

**Figure 2. Cascade model for the primary children with shared risk**

**variables freed; \* $p < .01$ , \*\* $p < .001$**



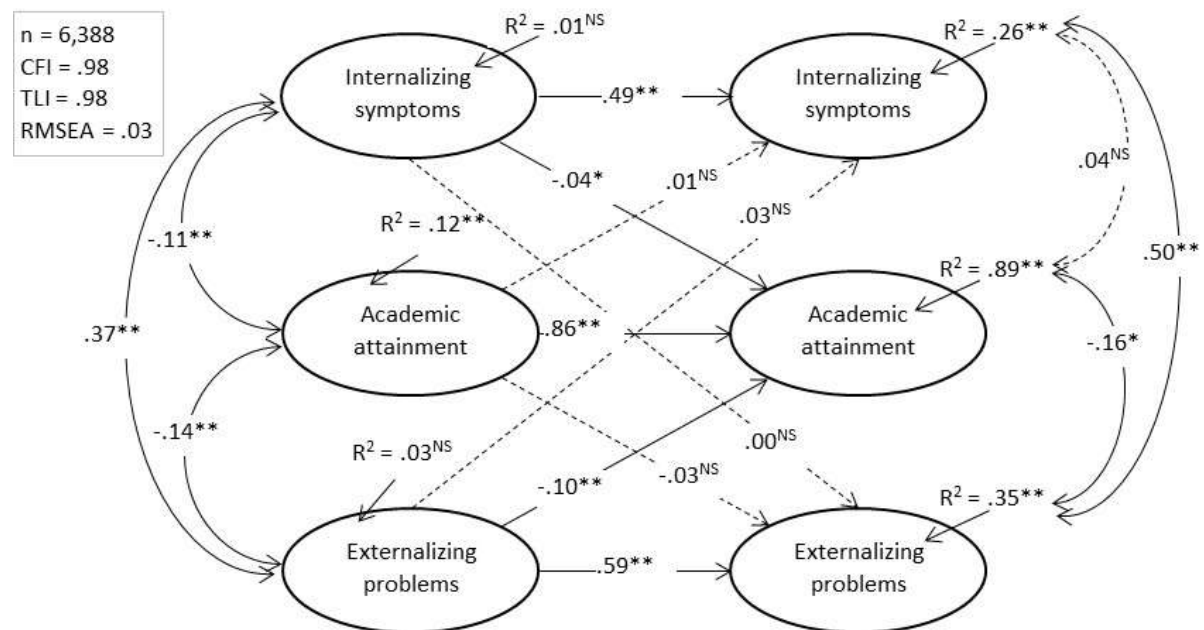
CFI: Comparative Fit Index; TLI: Tucker-Lewis Index; RMSEA: Root Mean Square Error of

Approximation; <sup>NS</sup> Not Significant; \*  $p < .01$ ; \*\*  $p < .001$

N.B. All latent variables are shown as ellipses; shared risk indicators were manifest variables.

# LONGITUDINAL PATHWAYS BETWEEN MENTAL HEALTH AND ACADEMIC PERFORMANCE

**Figure 3. Cascade model for the secondary school children with shared risk factors freed; \* $p < .05$ , \*\* $p < .01$ , \*\*\* $p < .001$**



CFI: Comparative Fit Index; TLI: Tucker-Lewis Index; RMSEA: Root Mean Square Error of

Approximation; <sup>NS</sup> Not Significant; \*  $p < .01$ ; \*\*  $p < .001$

N.B. All latent variables are shown as ellipses; shared risk indicators were manifest variables.

LONGITUDINAL PATHWAYS BETWEEN MENTAL HEALTH AND  
ACADEMIC PERFORMANCE

**Appendix 1: Me and My Feelings Questionnaire**

Emotional difficulties subscale (internalizing symptoms):

I feel lonely

I cry a lot

I am unhappy

Nobody likes me

I worry a lot

I have problems sleeping

I wake up in the night

I am shy

I feel scared

I worry when I am at school

Behavioural difficulties subscale (externalizing problems):

I get very angry

I lose my temper

I hit out when I am angry

I do things to hurt people

I am calm (reversed scored)

I break things on purpose

Response options: never (0), sometimes (1), always (2)